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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/402,820	10/12/1999	DANIEL G. CHAIN	20555/1203301-US1	6495

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EXAMINER	
DUFFY, PATRICIA ANN	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	09/402,820	CHAIN, DANIEL G.	
	Examiner	Art Unit	
	Patricia A. Duffy	1645	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *4-3-07*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *14,23,24,33,35 and 38* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *14,23,24,33,35 and 38* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *2006, 2007*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

RESPONSE TO AMENDMENT

The amendment filed 4-3-07 has been entered into the record. Claims 1-13, 15-22, 25-32, 34 and 36-37 have been cancelled. Claims 14, 23, 24, 33, 35 and 38 are pending and under examination.

The information disclosure statements filed 4-4-07 and 10-26-06 have been considered. Initialed copies are enclosed.

The text of Title 35 of the U.S. Code not reiterated herein can be found in the previous office action.

Rejections Withdrawn

All of the previous art rejections of record as withdrawn in favor of the new grounds of rejection set forth below based on amendments to the claims to recite that the amyloid beta peptide be "soluble in cerebrospinal fluid'.

The rejection of claims 14, 23, 24, 33 and 35 under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention is witdrawn in view of the amendment to the claims.

New Rejections Based on Amendment

Claims 14, and 24 are rejected under 35 U.S.C. 103(a) as being unpatentable over Saido et al (The Journal of Biochemistry, 269(21):15253-15257, 1994, Fee-based IDS Nov 16, 2001) in view of Takeda Chemical Industries Ltd., (EP 0 683 234 A1, published November 22, 1995, reference AC on PTOL-1449 filed 12 October 1999), Vigo-Pelfrey et al (Journal of Neurochemistry, 61:1965-1968, 1993) and Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97).

The claim is drawn to a monoclonal antibody that is free-end specific for the free n-terminus of an amyloid beta peptide binds to said free N-terminus said free terminus

and does not bind to the amyloid beta-precursor protein from which said amyloid beta peptide may be proteolytically derived wherein the amyloid beta peptide is soluble in cerebrospinal fluid.

Saido et al teach a polyclonal antibody 9204, that was produced using a synthetic heaxamer peptide DAEFRC (Asp-Ala-Glu-Phe-Arg-Cys) conjugated to keyhole limpet hemocyanin. The antibody distinguished the fragments possessing the exact amino terminus of AB from the intact precursors and other fragments including the secretase products. Antibody 9204 also recognized synthetic AB1-40 peptide but not AB2-40 peptide. Furthermore, Saido et al teaches that binding of antibody 9204 to AFF-C100 was inhibited by the haptenic peptide DAEFRC, but not by MADEFTC or by AEFRHC. Saido et al teaches that this indicates that the antibody has strict specificity toward the cleavage site with an accuracy of 1 amino acid residue (i.e. the instant free-end specific N-terminal specific). Saido et al teaches that the use of the cleavage site specific antibody provides for better relative quantitativeness. (see page 15254-55, column 1, Resultes, first and second paragraphs). Saido et al teaches that "similar approaches for producing the proteolytic product specific antibodies will be applicable to resolving the differential carboxyl-terminal processing of AB peptides...". Saido et al differs by not teaching a monoclonal antibody with the properties of polyclonal antibody 9204.

Takeda teaches that monoclonal antibodies that are specific for the N-terminal and C-terminal of AB are useful for the detection of AB1-40 and AB1-42 for the detection of AB species *in vitro* (see page 5) Takeda teaches that AB1-40 is water soluble (page 4, lines 33-41). Takeda teach the N and C-terminal peptide sequence of AB1-40.

Vigo-Pelfrey et al teach the specific structures of beta amyloid peptides from human cerebrospinal fluid (CSF). Vigo-Pelfrey et al teach that amino acid sequencing reveals species of amyloid beta with N-termini of Asp1, Glu3, His6, Glu11 and Val12. Laser desprption mass spectrometry confirmed the presence of amyloid beta species containing 27, 28, 30, 34, 35, 40, 42 and 43 amino acids all beginning at Asp1. Vigo-Pelfrey et al is

seen to teach the soluble amyloid beta species present in human CSF. Vigo-Pelfrey et al is seen to teach the free end C-termins terminus of AB1-40 is present and soluble in CSF.

Goding teaches routine methods of making monoclonal antibodies with defined immunogens.

It would have been *prima facie* obvious to one having ordinary skill in the art at the time that the invention was made to use the teachings of Saido et al to generate free-end N-terminal specific antibodies that do not bind the prescursor and bind species of soluble amyloid beta found in human cerebrospinal fluid using the conventional techniques of Goding et al because of the well established advantages of high-affinity, high specificity and unlimited supply that are central to monoclonal antibodies. One would have been motivated to make monoclonal antibodies to decrease the lot to lot variability that can happen with polyclonal antisera and Takeda et al teach that the monoclonal antibodies are useful for the detection of AB1-40 and AB1-42 for the detection of AB species *in vitro* and that AB1-40 is water soluble and present in the human cereprospinal fluid. One of ordinary skill in the art would have a reasonable expectation of success given the demonstrated immunogenicity of the epitope.

Claims 14 and 33 are rejected under 35 U.S.C. 103(a) as being unpatentable over Takeda Chemical Industries Ltd., (EP 0 683 234 A1, published November 22, 1995, reference AC on PTOL-1449 filed 12 October 1999) in view of Saido et al (The Journal of Biochemistry, 269(21):15253-15257, 1994, Fee-based IDS Nov 16, 2001; hereinafter Saido A), Saido et al (The Journal of Biological Chemistry, 268(33):25239-25243, 1993; herein after Saido B), Vigo-Pelfrey et al (Journal of Neurochemistry, 61:1965-1968, 1993) and Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97).

The claims are drawn to a monoclonal antibody that is free-end specific for the free C-terminus of an amyloid beta peptide 1-40 wherein the antibody binds to said free C-terminus said free terminus and does not bind to the amyloid beta-precursor protein

from which said amyloid beta peptide may be proteolytically derived wherein the amyloid beta peptide is soluble in cerebrospinal fluid.

Takeda teaches that monoclonal antibodies that are specific for the N-terminal and C-terminal of AB petides are useful for the detection of AB1-40 and AB1-42 for the detection of AB species *in vitro* (see pages 4-5). Takeda teaches that AB1-40 is water soluble (page 4, lines 33-41). Takeda teach the N and C-terminal peptide sequence of AB1-40. Takeda teach the monoclonal antibody BA-27a, that was considered to be specific for the C-terminus of beta amyloid (1-40), and weakly cross-reacted to beta-amyloid (1-38), (1-39) and beta amyoid (1-42) with a cross reactivity with 2% or less (page 34, lines 41-46). Takeda et al differs by not teaching a monoclonal antibody that has no cross-reactivity as "uniquely recognizes" the free C-terminal of AB1-40 and does not recognize the precursor.

Saido A teaches a polyclonal antibody 9204, that was produced using a synthetic heaxamer peptide DAEFRC (Asp-Ala-Glu-Phe-Arg-Cys) conjugated to keyhole limpet hemocyanin for the N-terminal of AB1-40. The antibody distinguished the fragments possessing the exact amino terminus of AB from the intact precursors and other fragments including the secretase products. Antibody 9204 also recognized synthetic AB1-40 peptide but not AB2-40 peptide. Furthermore, Saido et al teaches that binding of antibody 9204 to AFF-C100 was inhibited by the haptenic peptide DAEFRC, but not by MADEFTC or by AEFRHC. Said o et al teaches that this indicates that the antibody has strict specificity toward the cleavage site with an accuracy of 1 amino acid residue (i.e. the instant free-end specific N-terminal specific). Saido et al teaches that the use of the cleavage site specific antibody provides for better relative quantitativeness. (see page 15254-55, column 1, Results, first and second paragraphs). Saido et al teaches that "similar approaches for producing the proteolytic product specific antibodies will be applicable to resolving the differential carboxyl-terminal processing of AB peptides...". Saido et al teach that their unique methodology for producing such proteolytic produce-

specific antibodies now seems to have general applicability (page 15254 (column 1, see first paragraph results section).

Saido B teaches a general technique for producing antibodies that specifically distinguish a proteolyzed form from a given intact form and are free-end specific.

Vigo-Pelfrey et al teach the specific structures of beta amyloid peptides from human cerebrospinal fluid (CSF). Vigo-Pelfrey et al teach that amino acid sequencing reveals species of amyloid beta with N-termini of Asp1, Glu3, His6, Glu11 and Val12. Laser desprption mass spectrometry confirmed the presence of amyloid beta species containing 27, 28, 30, 34, 35, 40, 42 and 43 amino acids all beginning at Asp1. Vigo-Pelfrey et al is seen to teach the soluble amyloid beta species present in human CSF. Vigo-Pelfrey et al is seen to teach the free end C-termins terminus of AB1-40 is present and soluble in CSF.

Goding teaches routine methods of making monoclonal antibodies with defined immunogens.

It would have been *prima facie* obvious to one having ordinary skill in the art at the time that the invention was made to use the teachings of the Saido A and B to generate free-end C-terminal specific AB1-40 monoclonal antibodies that do not bind the prescursor using the conventional end-peptide immunization techniques of Saido A and B combined with monoclonal antibody technology of Goding et al because of the well established advantages of high-affinity, high specificity and unlimited supply that are central to monoclonal antibodies and Takeda et al teach that antibodies with high sensitivity and specificity for amyloid peptide, including AB1-40 are desired and Vigo-Pelfrey teaches that AB1-40 speices are soluble in CSF. One would have been motivated to make screen for free-end specific monoclonal antibodies to eliminate the residual cross-reactivity of the monoclonal antibody BA-27(a) of Takeda and because Takeda et al teach that the prior art assays lack sensitivity and specificity and that highly specific monoclonal antibodies are useful for the detection of AB1-40 and AB1-42 species *in vitro* and that unique antibodies would reduce the background and increase the sensitivity of the immunoassay

for AB1-40. One of ordinary skill in the art would have a reasonable expectation of success given the demonstrated immunogenicity of the C-terminal epitope of AB1-40 as shown by Takeda and the success of Saido A for the N-terminal epitope and that Saido A teaches that similar approaches will be applicable to resolving the differential carboxy terminal processing of AB peptides.

Claims 23 and 35 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Saido et al (The Journal of Biochemistry, 269(21):15253-15257, 1994, Fee IDS Nov 16, 2001), Takeda Chemical Industries Ltd., (EP 0 683 234 A1, published November 22, 1995, reference AC on PTOL-1449 filed 12 October 1999), Vigo-Pelfrey et al (Journal of Neurochemistry, 61:1965-1968, 1993) and Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97) as applied to claim 14 and 24 above and further in view of Seubert et al (U.S. Patent 6,114,133, issued September 5, 2000 and filed November 14, 1994) and Duenas et al (BioTechniques, 16(3):476-483, 1994) for reasons made of record in the Office Actions mailed 4-22-03, 1-26-06 and herein.

The claims are drawn to single chain antibodies that are free-N-terminal specific for AB peptide soluble in cerebrospinal fluid.

The teachings for Saido et al (The Journal of Biochemistry, 269(21):15253-15257, 1994, Fee-based IDS Nov 16, 2001) in view of Takeda Chemical Industries Ltd., (EP 0 683 234 A1, published November 22, 1995, reference AC on PTOL-1449 filed 12 October 1999), Vigo-Pelfrey et al (Journal of Neurochemistry, 61:1965-1968, 1993) and Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97) as combined are set forth supra. The references as combined fail to teach single chain antibodies.

Seubert et al teaches the use of antibodies that bind AB peptides in *in vitro* or *in vivo* assays that screen for inhibitors of AB peptide formation (see columns 4-5, Summary of the Invention). Seubert et al teach that in addition to monoclonal antibodies, "... the detection techniques of the present invention will also be able to use antibody fragments,

such as F(ab), Fv, VL, VH, and other fragments." Seubert et al also teach that "It would also be possible to employ recombinantly produced antibodies (immunoglobulins) and variation thereof as now well described in the patent and scientific literature. See, for example EPO 8430268.0; EPO 85102665.8; EPO 85305604.2; PCT/GB 85/00392; EPO 85115311.4; PCT/US 86/002269; and Japanese application 85239543." (see column 10, first full pargraph).

Duenas et al teach art accepted conventional methods of intra- and extracellular expression of a single chain Fv antibody fragment (scFv) in *E. coli*. Duenas et al teach that cloning of immunoglobulin variable regions and bacterial expression of antibody fragments was routinely performed in the art at the time that this invention was made (see page 476, column 2, Introduction).

It would have been *prima facie* obvious to one having ordinary skill in the art at the time that the invention was made to modify the free-end, N-terminal specific monoclonal antibody according to the combination of Saido et al (The Journal of Biochemistry, 269(21):15253-15257, 1994, Fee-based IDS Nov 16, 2001) in view of Takeda Chemical Industries Ltd., (EP 0 683 234 A1, published November 22, 1995, reference AC on PTOL-1449 filed 12 October 1999) and Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97) supra, by means of expression as a single chain Fv antibody fragment (scFv) according to the vectors and methodology of Duenas et al because Seubert et al teach that Fv and other antibody fragments including those that have been recombinantly produce that bind AB peptides are useful in a variety of detection techniques for use in screening or diagnostic assays.

Claims 23 and 38 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Takeda Chemical Industries Ltd., (EP 0 683 234 A1, published November 22, 1995, reference AC on PTOL-1449 filed 12 October 1999),Saido et al (The Journal of Biochemistry, 269(21):15253-15257, 1994, Fee-based IDS Nov 16, 2001; hereinafter

Saido A), Saido et al (The Journal of Biological Chemistry, 268(33):25239-25243, 1993; herein after Saido B), Vigo-Pelfrey et al (Journal of Neurochemistry, 61:1965-1968, 1993) and Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97) as applied to claim 14 and 33 above and further in view of Seubert et al (U.S. Patent 6,114,133, issued September 5, 2000 and filed November 14, 1994) and Duenas et al (BioTechniques, 16(3):476-483, 1994).

The teachings of Takeda Chemical Industries Ltd., (EP 0 683 234 A1, published November 22, 1995, reference AC on PTOL-1449 filed 12 October 1999) in view of Saido et al (The Journal of Biochemistry, 269(21):15253-15257, 1994, Fee-based IDS Nov 16, 2001; hereinafter Saido A), Saido et al (The Journal fo Biological Chemistry, 268(33):25239-25243, 1993; herein after Saido B), Vigo-Pelfrey et al (Journal of Neurochemistry, 61:1965-1968, 1993) and Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97) are set forth above. The references as combined differ by not teaching single chain antibodies.

Seubert et al teaches the use of antibodies that bind AB peptides in *in vitro* or *in vivo* assays that screen for inhibitors of AB peptide formation (see columns 4-5, Summary of the Invention). Seubert et al teach that in addition to monoclonal antibodies, "... the detection techniques of the present invention will also be able to use antibody fragments, such as F(ab), Fv, VL, VH, and other fragments." Seubert et al also teach that "It would also be possible to employ recombinantly produced antibodies (immunoglobulins) and variation thereof as now well described in the patent and scientific literature. See, for example EPO 8430268.0; EPO 85102665.8; EPO 85305604.2; PCT/GB 85/00392; EPO 85115311.4; PCT/US 86/002269; and Japanese application 85239543." (see column 10, first full pargraph).

Duenas et al teach art accepted conventional methods of intra- and extracellular expression of a single chain Fv antibody fragment (scFv) in *E. coli*. Duenas et al teach that cloning of immunoglobulin variable regions and bacterial expression of antibody fragments

was routinely performed in the art at the time that this invention was made (see page 476, column 2, Introduction).

It would have been *prima facie* obvious to one having ordinary skill in the art at the time that the invention was made to modify the free-end, C-terminal specific monoclonal antibody that binds a soluble CSF amyloid beta peptide according to the combination of Takeda Chemical Industries Ltd., (EP 0 683 234 A1, published November 22, 1995, reference AC on PTOL-1449 filed 12 October 1999) in view of Saido et al (The Journal of Biochemistry, 269(21):15253-15257, 1994, Fee-based IDS Nov 16, 2001; hereinafter Saido A), Saido et al (The Journal fo Biological Chemistry, 268(33):25239-25243, 1993; herein after Saido B), Vigo-Pelfrey et al (Journal of Neurochemistry, 61:1965-1968, 1993) and Goding (Monoclonal Antibodies, Academic Press Inc., London 1983, pages 56-97) above, by means of expression as a single chain Fv antibody fragment (scFv) according to the vectors and methodology of Duenas et al because Seubert et al teach that Fv and other antibody fragments including those that have been recombinantly produce that bind AB peptides are useful in a variety of detection techniques for use in screening or diagnostic assays.

Response to Arguments

Applicant's arguments have been carefully considered but are not persuasive. Applicants argue that the references do not teach that the amyloid beta peptide of the prior art is soluble in CSF or detection in the CSF. This is not persuasive because Vigo-Pelfry et al of record establishes that the amyloid beta species of the prior art as combined are soluble in CSF as they are not removed by a centrifugation process. Applicants argue that there is no suggestion or evidence that the antibodies would bind the peptide in CSF. This is not persuasive, since the same peptide is present in CSF and the antibody would be expected to bind the peptide present in CSF. The peptides detected by ELISA or western blotting or other competition experiments of the art have

been demonstrated by the prior art to be present in human cerebrospinal fluid. Applicants argue that there is no suggestion that the antibodies be used to detect peptides in the CSF. This is not persuasive, the claims are not drawn to a method of detection of peptides in the CSF, but merely that the antibodies bind peptides soluble in the CSF and Vigo-Pelfrey et al teach that the peptides of the prior art are in fact present in the CSF. Applicants essentially argue that there is no suggestion in the art as combined to make the monoclonal antibodies. This is simply not so, the motivation has been articulated and the amyloid peptides of the art are demonstrated by the prior art to be present in human cerebrospinal fluid. Applicants argue that Saido's methodology is not reproducible and provides no reasonable expectation of success. This files in the face of the teaching of Saido that teaches routine methodology to make the antibodies. The methods of Saido are conventional. Mere routine screening is required. That the screening may be extensive is not a consideration, because extensive screening by automation is routine in the antibody arts. Expectation of success is provided because a monoclonal antibody could be generated and therefore its presence provides an expectation of success of producing others like it. In *In re Wands*, 858 F.2d 731, 8 USPQ2d 1400 (Fed. Cir. 1988), the court reversed the rejection for lack of enablement under 35 U.S.C. 112, first paragraph, concluding that undue experimentation would not be required to practice the invention. The nature of monoclonal antibody technology is such that experiments first involve the entire attempt to make monoclonal hybridomas to determine which ones secrete antibody with the desired characteristics. The court found that the specification provided considerable direction and guidance on how to practice the claimed invention and presented working examples, that all of the methods needed to practice the invention were well known, and that there was a high level of skill in the art at the time the application was filed. Furthermore, the applicant carried out the entire procedure for making a monoclonal antibody. As such, the prior art is enabled for making the monoclonal and single chain antibodies. This is the same issue here, there is direction and guidance

and motivation in the prior art. The courts have held, in contrast to Applicants statement that "The motivation need not be found in the references sought to be combined, but may be found in any number of sources, including common knowledge, the prior art as a whole, or the nature of the problem itself." *In re Dembiczak*, 175 F.3d 994, 999 (Fed. Cir. 1999). As we explained in *Motorola, Inc. v. Interdigital Tech. Corp.*, 121 F.3d 1461, 1472 (Fed. Cir. 1997), "there is no requirement that the prior art contain an express suggestion to combine known elements to achieve the claimed invention. Rather, the suggestion to combine may come from the prior art, as filtered through the knowledge of one skilled in the art." *DyStar Textilfarben GmbH & Co. Deutschland KG v. C.H. Patrick Co.*, 80 USPQ2d 1641 (Fed. Cir. 2006). Futhermore, the ability to generate antibodies to neo-antigenic determinants created by proteolysis was known as early as 1991 (Abbas et al, Cellular and Molecular Immunology, WB Saunders Company, 1991, page 52). Applicant's arguments that the technology is not combinable is inconsistent with the skill in the art and the teachings and art of record. The peptides of the prior art to which the monoclonal and single chain antibody bind are found soluble in CSF as clearly demonstrated by Vigo-Pelfry.

Status of Claims

Claims 14, 23, 24, 33, 35, and 38 stand rejected.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL.** See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not

mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Patricia A. Duffy whose telephone number is 571-272-0855. The examiner can generally be reached on M-Th 6:30 am - 6:00 pm. If attempts to reach the examiner by telephone are unsuccessful, the examiner's Supervisor, Jeffrey Siew can be reached on 571-272-0787.

The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Patricia A. Duffy
Primary Examiner
Art Unit 1645